PROCEEDINGS

1990 MANITOBA BEEF CATTLE SEMINAR FEBRUARY 13th

MANITOBA AGRICULTURE

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MANITOBA BEEF CATTLE SEMINAR

February 13, 1990
Agricultural Extension Centre
Brandon, Manitoba

Our annual BEEF SEMINAR is trying a new format this year – a one day session in February. Join us for the latest ideas on calving management and trace minerals nutrition.

AGENDA

MORNING SESSION

Chairperson: Dr. Guenther Rahnefeld, Agriculture Canada, Brandon

8:30 a.m.	Registration and Coffee – Visit Displays
9:45	Opening Remarks
10:00	**Factors Relating to Calving Difficulties** – Dr. Bob Bellows – An in-depth review of the many factors affecting calving, such as management, nutrition, cow condition, pelvic measurements, and more.
11:30	**Questions and Answers**
12:00	Noon Lunch

AFTERNOON SESSION

Chairperson: Norm Hemstad, Livestock Specialist, Manitoba Agriculture, Brandon

1:00 p.m.	**Manitoba Trace Mineral Studies** – Lyle McNichol – An update on the latest trace mineral findings in Manitoba
1:30	**Trace Mineral Interaction** – Dr. Karin Wittenberg – A look at how minerals affect each other, what levels and ratios should be in your rations and a look at how high water sulfates affect copper utilization
2:30	Coffee
3:00	**Trace Mineral Problems** – Dr. Jim Dyck – A vet looks at identifying and treating trace mineral problems.
4:00	**Questions and Answers**
4:30	Adjourn

We hope that you enjoy the 1990 Manitoba Beef Cattle Seminar. I would like to thank the Planning Committee of Dr. Guenther Rahnefeld, Agriculture Canada, and Norm Hemstad, Manitoba Agriculture for their assistance with this program.

See you in '91!

Keith Levenick, Principal
Agricultural Extension Centre

SEMINAR SPEAKERS

DR. BOB BELLOWS - A cattleman's researcher from the Livestock and Range Management Centre, Miles City, Montana - Dr. Bellows brings a practical approach, as well as quality results to the whole calving problem area.

DR. JIM DYCK - As one of the veterinarians with the Swan Valley Vet. Clinic in Swan River, Dr. Dyck has been treating cattle for and taking part in mineral studies since the mid 70's. They are especially concerned with copper and selenium problems.

LYLE McNICHOL - As the Livestock Specialist for Manitoba, in the Northwest Region since '74, Lyle has been involved in many of the trace mineral studies and treatment trials that have been done in that region.

DR. KARIN WITTENBERG - A ruminant nutritionist with the Animal Science Department of the University of Manitoba - Dr. Wittenberg has been working with trace minerals for the past five years. She has been particularly interested in molybdenum uptake and transfer in cow-calf operations but has also done some work with feedlot animals.

DYSTOCIA IN BEEF CATTLE

R.A. BELLOWS

USDA - ARS
Fort Keogh Livestock and Range Research Laboratory
Miles City, MT[a]

Introduction

Calf deaths at or shortly after calving result in losses of over 3.5 million calves annually in the United States with a value exceeding $850 million. Approximately 45% of these losses are caused by dystocia (defined as delayed and/or difficult parturition) accounting for a $380 million loss due to this single factor. Dystocia results in additional losses because dams experiencing dystocia exhibit depressed rebreeding rates in the breeding season immediately following dystocia and severe dystocia results in reduced growth rates in the affected calf. Thus, total losses from dystocia are conservatively estimated to exceed $750 million annually.

Factors associated with dystocia in cattle have been reviewed by Price and Wiltbank (1978a). They conclude that calf size, calf sex, gestation length, breed, inbreeding, genotype, nutrition and age, parity and pelvic area of the dam are the most important factors affecting dystocia.

This paper will briefly discuss two of these factors - - genetics and nutrition.

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[a] USDA-ARS and Montana Agric. Exp. Sta., Cooperating. Contribution No. J-2391 from Montana Agric. Exp. Sta.

Genetic Factors

The selection of cattle for growth and efficient meat and milk production has essentially been practiced since cattle were first domesticated. Of course, selection for color, size, shape, ear length, horn length, etc. have caused some interesting and sometimes disastrous trips down sideroads. However, it is clear that cattle emerging today as productive, desirable and potentially profitable cattle are those that produce a desirable product efficiently. At the present time, we, as an industry, are trying to determine what is a desirable product and since there are many differing opinions, cattlemen find themselves shooting at a moving target. We must predict on the basis of often incomplete data what the demand will be 5 to 10 or more years down the road. Not an easy task.

Early selection and breeding programs were based on experience, opinions and a goodly amount of guesswork and bias. Work at the Miles City Research Station laid the foundation for research and data that has taken some of the guesswork out of prediction of productivity. The first research on record of performance (ROP), progeny testing and some of the first work on crossbreeding started at Miles City in the 1930's. With results from these studies, producers and scientists were given quantitative estimates for heritabilities and genetic correlations for economically important traits. These numerical values, when combined with modern computational capabilities, have taken animal breeding out of the guesswork mode and into the

realm of prediction of outcome from various matings or mating systems. Accuracy of these predictions is not perfect but is improving as more data become available.

Individuals associated with the cattle industry, whether consultants, producers or scientists have a wealth of information available regarding breed differences in productivity -- birth, weaning and yearling weights, calving ease, scrotal circumference, hip height, milk production and maternal effects. Results from numerous research studies are available that show productivity of many breeds under various environments.

Other publications are available showing performance values of numerous sires for the traits of economic importance. In addition, not only are the figures available for a sire, but performance of his daughters are also available. These values are Expected Progeny Differences (EPD) and provide comparisons between animals within a given breed. It is not the purpose of this paper to give a review of breed differences or a description of use of EPD, but these publications and summaries must be recognized as the best tools available to effectively change the genetics of a herd in any direction desired. This information is of major value when we consider just how is the best way to reach the goal of hitting that moving target mentioned earlier.

At the present time, consumers are sending the message they want beef that is consistent in quality, with low visible fat, documentation of caloric and cholesterol content, with assurance the product is wholesome and free from drug residues. There are other preferences, too, such as price and size of cut, but all are important. Gosey (1987) states, "Fortunately

there is not one consumer with one rigid set of specifications, but a variety of individual preferences to be met." This means that there is demand for a wide range of product in our beef market.

These demands from the consumer move back through the production chain and eventually impact the producer. At this time, emphasis is being placed on matching cow size, milk production and reproduction to the feed and management environment. Production systems available involve purebred matings or various forms of crossbreeding with emphasis on production of leaner, heavier muscled and, in some instances, larger cattle and calves. The combination of these recommendations and trends could potentially encourage breeding moderate-size cows to terminal sires with rapid growth rates, both within or among breeds. The reason for this is shown in Table 1 indicating the larger sire breeds have the potential of producing offspring with desirable carcasses.

However, these same breeds have higher average calf birth weights resulting in an increase in difficult births (Figure 1). This relationship represents one of the major "tradeoffs" that must be carefully managed within the beef breeding-production system.

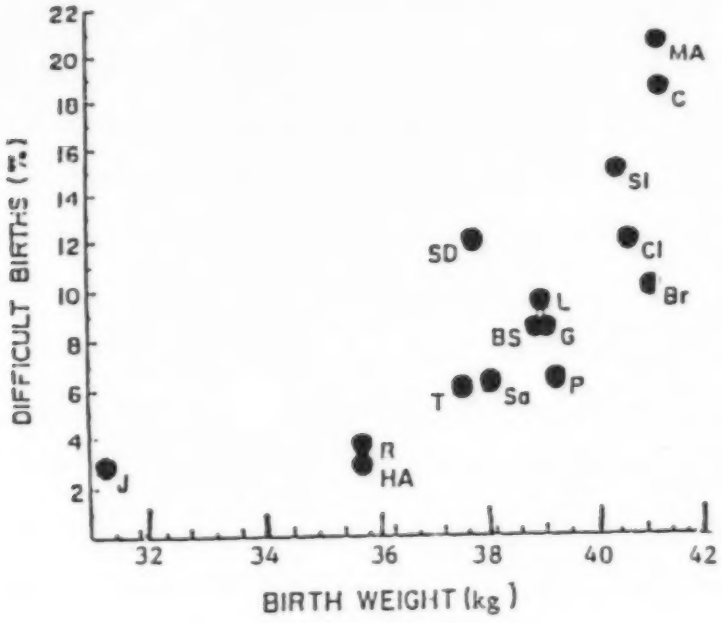
Heritabilities and genetic correlation. Today's informed cattlemen and their advisors are well aware of numerical values for heritabilities and genetic correlations. Heritability is defined as the ratio of additive genetic variation to total variation in a trait. The numerical

Table 1. Breed crosses grouped into seven biological types on the basis of four major criteria (Cundiff, 1986)[a]

Breed group	Growth rate & mature size	Lean to fat ratio	Age at puberty	Milk production
Jersey (J)	X	X	X	XXXXX
Hereford-Angus (HA)	XX	XX	XXX	XX
Red Poll (Rp)	XX	XX	XX	XXX
Devon (D)	XX	XX	XXX	XX
South Devon (Sd)	XXX	XXX	XX	XXX
Tarentaise	XXX	XXX	XX	XXX
Pinzgauer (P)	XXX	XXX	XX	XXX
Brangus (Bn)	XXX	XX	XXXX	XX
Santa Gertrudis (Sg)	XXX	XX	XXXX	XX
Sahiwal (Sw)	XX	XXX	XXXXX	XXX
Brahman (Bm)	XXXX	XXX	XXXXX	XXX
Brown Swiss (B)	XXXX	XXXX	XX	XXXX
Gelbvieh (G)	XXXX	XXXX	XX	XXXX
Holstein (Ho)	XXXX	XXXX	XX	XXXXXX
Simmental (S)	XXXXX	XXXX	XXX	XXXX
Maine-Anjou (M)	XXXXX	XXXX	XXX	XXX
Limousin (L)	XXX	XXXXX	XXXX	X
Charolais (L)	XXXXX	XXXXX	XXXX	X
Chianina (Ci)	XXXXX	XXXXX	XXXX	X

[a] Increasing number of X's indicate relatively higher levels of performance and older age at puberty.

Figure 1. Breed of sire of calf averages for birth weight (X) and difficult births (Y) for Hereford and Angus females calving at 4 years of age and older (Cundiff, 1986).



See Table 1 for breed designations.

Table 2. Pelvic area:birth weight factors for predicting dystocia (modified from Deutscher, 1988)

Heifer			PA:BW		Estimated unassisted birth weight	
Age (mo)	Weight, kg	PA (cm^2)	Lb.	Kg	Lb.	Kg
12-14	249-317	120-160	2.2	4.7	60-73	27-33
18-19	317-385	160-200	2.7	5.8	64-74	29-74
23-24	363-431	200-240	3.3	7.2	65-73	29-33

value is a measure of genetic likeness between a sire and his offspring. Genetic correlations are a measure of the relationship between two traits that arise from common genes that influence the trait. For example, the genetic correlation between birth weight and postweaning growth is moderate to high and is positive. This means the correlation is important, and there are common genes (or possibly the same genes) controlling or affecting both traits. This relationship is important because it is telling us that as we select animals for high weaning weights and rapid growth rate in the feedlot we are selecting for the same gene (s) that result in more rapid growth rate before birth, and this results in high birth weights which are accompanied by more calving difficulty. This does not present an unmanageable problem, but it means these relationships must be evaluated carefully or the production system will suffer because of a high dystocia rate and potentially an increase in calf deaths.

These can be managed by looking closely at EPD values available on many of the sires available for use in the industry. By carefully studying and using these values, the producer can manage the tradeoffs that exist and change the genetic makeup of the herd in the desired directions.

Previous studies (Bellows et al., 1971; Price and Wiltbank, 1978b) have shown that pelvic area of the dam at calving is negatively associated with dystocia. This relationship means that dams with larger pelvic areas have a greater probability of calving without difficulty.

The relationships between pelvic area in cows and bulls and the possibility of selecting for larger pelvic areas to reduce dystocia have been reviewed by Brinks (1987) and Deutscher (1989). These reviews conclude that increasing pelvic area of the dam while holding birth weight constant will reduce dystocia; selection for larger pelvic areas in bulls should result in a correlated increase in pelvic size of female offspring and that measuring pelvic size in heifers rather than relying solely on weight and skeletal size appears justified. Methods of increasing pelvic area include culling -- selection of the heifer, breeding for larger pelvic areas and feeding the heifer during replacement development so that the genetic potential for skeletal growth -- including pelvic size can be fully realized.

When an analysis is made of the most important factors affecting dystocia, the literature supports calf birth weight, calf sex and pelvic area of the dam as being the most important factors identified. It is interesting to note that calf sex and the genetic potential for the calf to grow in utero are genetically determined and are established at conception. Also, the relative pelvic size at calving exists at conception. Thus, a major portion of the dystocia problems are in place at conception and little can be done to alter the end result. This scenario points out the importance of wise sire selection -- holding growth rate to a moderate level in calves from first-calf heifers and feeding the heifer adequately so skeletal development is not retarded. It also

suggests that sex control in calves from first-calf heifers (produce all females) would be a valuable tool in combating dystocia.

Nutrition Factors

The effects of nutrition on dystocia must include a discussion of nutrition effects on development of the replacement heifer. Many studies have shown marked effects of postweaning-prebreeding nutritional regime on heifer puberty, but some studies have clearly shown that other effects are also involved (Price and Wiltbank, 1978a; Dzuik and Bellows, 1983; Meijering, 1984). These additional effects have included measurable differences in yearling, 18-month and precalving pelvic measurements of the heifer. Feed levels that allow maximum growth of the pelvic opening result in pelvic dimensions 2 to 8% greater and a decreased incidence of calving difficulty up to 10%. Price and Wiltbank (1978a) concluded that a constant level of dystocia can be maintained only if pelvic area increases in proportion to calf size.

Deutscher (1989) reported a prebreeding ratio of pelvic area; calf birth weight of 4.7 cm^2/lb. was found to be the pivotal point between calving assistance needed and no assistance. He suggests obtaining pelvic measurements before breeding and dividing that area by a factor to estimate the calf birth weight the heifer can deliver as a 2 yr. old without severe difficulty. These have been published and are summarized in Table 2.

In summarizing the data from Short et al. (1979), we found precalving pelvic areas of 210 to 250 cm^2 in 23- to 24-month old heifers weighing 800 to 975 lb. (363 to 442 kg). The pelvic area could be divided by 3.8 for lb. or 8.4 for kg to give a calf birth weight range of 57 to 75 lb. or 26 to 34 kg for an estimated birth weight of less than 50% assistance. Thus, our data are less optimistic than the predictions from Deutscher.

The last point to be covered on pelvic area is that of selection of animals for larger pelvic dimensions. Values reported indicate an average heritability of .54 for pelvic area (Deutscher, 1989). This value is high enough to indicate pelvic area is a trait that could be altered by selection. However, there are some things that we must consider. The genetic correlation between pelvic area and calf birth weight is positive and values as high as .73 have been reported (Benyshek and Little, 1982). By taking average heritability and genetic correlation values from the literature and making a number of basic assumptions, the following effects can be calculated: a 10% increase in pelvic area would theoretically be accompanied by a .02 drop in CD score (desirable) and a .9-kg (1.98 lb.) increase in calf birth weight (undesirable?). Why is it? Because pelvic area is another growth trait and selection for growth traits will increase calf birth weight. Unfortunately, we do not have research studies on this specific area of selection.

The next factor to be considered is the effect of nutrition on calf birth weight, and this cannot be separated

from discussion of its effect on postpartum reproduction of the dam.

Price and Wiltbank (1978a) and Bellows (1984) reviewed literature describing effects of nutrition on dystocia. They concluded that gestation feed level of the dam can affect birth weight. However, the effect on dystocia is not consistent and cannot be considered predictable. The exceptions to these conclusions are in instances of very low or very high feed levels. Calving patterns and behaviour are affected in these animals, seemingly, because dams and calves on very low levels of feeding are weak and less active during and after calving. High levels of feeding result in a fat-filled birth canal, which effectively reduces the size of the opening with the same effect as a small pelvic area. The review by Bellows (1984) is shown in Table 3. In addition to the effects summarized above, there is a trend for more dystocia to occur in dams on low feed levels, and low feed levels increased the incidence of scours and depressed calf survival at weaning. Another dramatic effect of low gestation feed levels is the depression of postpartum reproduction of the dam with both estrus and pregnancy rates being affected.

One must conclude that even though gestation feed levels can alter birth weights, low feed levels are not recommended because: 1) a lack of significant and predictable effects on incidence of dystocia; 2) the consistent depressing effects on calf survival and subsequent reproduction in the dam. Likewise, very high feed levels must be avoided

Table 3. Some effects of gestation feed level (modified from Bellows, 1984)

Data source	Precalve feed level	Precalv. pelvic area, (cm^2)	Calf birth wt. (kg)	Dystocia (%)	Calf survival: At birth (%)	Calf survival: At wean. (%)	Scours incidence: Affected (%)	Scours incidence: Mortality (%)	Subsequent reproduction: Estrus (%)	Subsequent reproduction: Preg. (%)
Laster, 1974	Low, 4.9 kg TDN	--	26	26	--	--	--	--	--	--
	Med., 6.2 kg TDN	--	28	17	--	--	--	--	--	--
	High, 1.7 kg TDN	--	29*	18	--	--	--	--	--	--
	90 d prepartum									
Corah et al., 1975										
Heifers	Low, 11.4 Mcal ME	--	28	28	90	--	--	--	56	--
	High, 17.6 Mcal ME	--	30*	27	97	--	--	--	74	--
	100 d prepartum									
Cows	Low, 8.4 Mcal DE	--	27	same	90	71	52	19	38	--
	High, 19.4 Mcal DE	--	35*	same	100	100	33	0	48	--
	100 or 30 d prepartum									
Dunn et al., 1969	Low, 7.3 Mcal ME	--	29	16	--	--	--	--	44	60
	High, 13.3 Mcal ME	--	32*	33	--	--	--	--	69*	68
	140 d prepartum									
Wiltbank and Rennenga, 1982	Low 7.3 Mcal ME	239	30	34	--	--	--	--	--	--
	High 13.3 Mcal ME	249	33*	36	--	--	--	--	--	--
	115 d prepartum									
Kroker and Cummins, 1979	Low, lost .5 kg/d	276	24	25	100	45	--	--	90	80
	Med., maintain wt.	261	29	5	89	75	--	--	80	35*
	High, gain .75 kg/d	311	33*	15	79	79	--	--	100	68
	90 d prepartum									
Bellows and Short, 1978	Low, 3.3 kg TDN	250	28	50	--	--	--	--	38	66
	High, 6.3 kg TDN	256	30*	47	--	--	--	--	67*	84*
	90 d prepartum									
Bellows et al., 1982	Low, 3.6 kg TDN	261	36	64	--	--	--	--	55	67
	High, 6.8 kg TDN	268	36	58	--	--	--	--	68	79

[a] Values represent values within studies cited.
* $P<.05$.

because (1) they result in increased calving problems because fat deposits in the birth canal reduce the size of calf that can be accommodated, and (2) excess feed levels are an unnecessary expense to the livestock operation.

Some studies have implicated dietary protein level during gestation as having effects on incidence and severity of dystocia. The literature is not consistent in this regard with three of the most recent studies summarized in Table 4. It can be seen that our 1978 work suggested an effect, but this was not confirmed by additional work in our laboratory (Anthony et al., 1986) or by scientists at Kansas State University (Bolze and Corah, 1988b). The only differences we could identify between the two studies at Miles City were higher body condition scores in the pregnant dams and higher environmental temperatures in 1986. What effects these differences caused are unknown. Inconsistent birth weight effects of maternal dietary protein during gestation have also been reported by Eimers et al. (1983). Thus, the conclusion reached is that the effects of gestation dietary protein are not consistent and, at our present state of knowledge, the effects are not predictable.

The role of minerals in the gestation diet affecting birth weight and dystocia is not clear. Few studies have been reported and any differences have been in relation to differences in birth weight. Dufty et al. (1977) reported plasma zinc levels of cows remained relatively constant until late pregnancy when a decline occurred. It was interesting

Table 4. Effect of gestation protein diet on birth weight and dystocia

	Protein content	
	Low (75-81% NRC)	High (140-150% NRC)
Bellows et al. (1978)		
Calf birth weight (kg)	33	38*
CD score	1.6	2.2
% assisted	42	58†
Anthony et al. (1986)		
Calf birth weight (lb.)	35	36
CD score	1.6	1.6
% assisted	36	36
Bolze & Corah (1988b)		
Calf Birth weight (lb.)	41	41
CD score	2.2	1.7
% assisted	48	37

* P<.05.
† P<.10.

Table 5. Zeranol implant effects (Bolze and Corah, 1988a)

	Treatment group						
	Fall calve Simmental			Spring calve Simmental		Fall calve Angus	
	C[a]	PW	W	C	W	C	W
No. head	49	49	49	46	46	66	68
Pelvic area (cm							
Yearling	194[e]	196[e]	207[f]	188[e]	195[f]	168[e]	175[f]
Precalve	272[e]	261[f]	268[e,f]	246[e]	233[f]	232[e]	236[e]
Calvings (%)							
Unassisted	81	79	86	60	56	57	64
C-section	5	--	--	3	4	--	--

[a] Implanted at: C = control; PW = preweaning, W = weaning.
[e,f] Different superscripts, P<.05.

that dams experiencing dystocia showed a greater decline than those without dystocia. However, Pryor (1976) reported no difference in plasma zinc levels between dams calving with or without dystocia. Pond et al. (1983) reported that calf birth weights were increased in zinc-supplemented dams in three studies conducted at MARC. No differences were reported in dystocia. These results could be interpreted to suggest that adequate calf growth and normal parturition are dependent on adequate mineral availability.

The last point to be discussed is not actually part of the assigned topic. That point is the use of growth-promoting implants to stimulate pelvic growth and, in turn, reduce dystocia. Results of published studies are somewhat inconsistent and two studies are summarized in Table 5 (Bolze and Corah, 1988a). They conclude that zeranol stimulated pelvic development between weaning and 1 yr. of age, and there was a residual effect evident at calving in one study. There was no effect on percentage of heifers requiring assistance at calving. Deutscher et al. (1986) reported that heifers receiving implants at either 6 or 9 mo. of age had larger precalving pelvic areas (+2% and +3% for 6- and 9- mo. implant groups, respectively), but no effect was noted on dystocia incidence.

Again, the literature is not in agreement and recent reports by Bohlender (1989) indicate use of Synovex-C implants given at branding result in both an increase in pelvic area at calving and a reduction in dystocia. A study is underway in Canada to determine effects of Synovex-C given at branding on pelvic area and dystocia. Results to date indicate a slight

reduction in pregnancy rate in the implanted heifers and an 11 cm^2 increase in pelvic area prebreeding. This increase, however, was not evident by mid-gestation or precalving. Dystocia data have not been summarized (L.M. Rutter, personal communication).

In my opinion, we must be careful in using growth stimulants to correct pelvic area-related dystocia problems. These compounds can mask genetic ability to grow and calve unassisted. It is also important to remember that females that calve unassisted because of the implant will have a greater probability of becoming a replacement female in the herd and will then be eligible to produce other females that would become replacements. What effects this could have on the overall assisted birth rate including the genetic aspects is unknown, but I am afraid it would not be good. This is particularly true if the implants were used repeatedly and then if the implants were removed from the market.

Finally, I firmly believe that heifers with dystocia scores of 3 or 4 are good cull prospects. We have data that indicate prepartum hormone levels in dams experiencing dystocia are not the same as those that do not experience dystocia. This implies a physiological difference between the two types of animals. Heifers that experience dystocia problems because of large birth weight or small pelvic area are also cull prospects because they are telling you that they do not fit your management and production system. Don't forget that over 50% of the heifers calved unassisted. Let them be your herd builders.

SUMMARY

Dystocia has a major economic impact resulting from calf deaths, reduced calf gain and poor subsequent reproductive performance of the affected dam. Dystocia results from genetic factors, and these are associated with genetic effects on growth -- growth of the pelvic area of the dam and growth of the fetus in utero. When fetus growth is disproportional to pelvic growth, an incompatability and dystocia result. Data are available from sire summaries on expected progeny difference (EPD values) for sire selection to move the genetics of the cow herd in essentially any direction desired. These can be used to hold birth weights down and to obtain high growth rates in the heifer so the pelvic opening is relatively large.

Nutrition effects (energy or protein) on dystocia are inconsistent with reported increases in birth weights from high levels of gestation feeding, but this does not result in predictable effects on dystocia. Excessively fat heifers experience high rates of dystocia and calf loss. The role of minerals in the gestation diet and dystocia is not clear. Some evidence is available indicating adequate calf growth and normal parturition are dependent on minerals being available in adequate amounts.

The use of growth stimulants to correct dystocia problems resulting from small pelvic areas should be used very judiciously. Repeated use could potentially leave a producer in a situation of reaping high rates of dystocia, especially if the products used are removed from the market.

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TRACE MINERALS IN MANITOBA

The occurrence of trace mineral deficiencies was first recognized in the early years around 1900 when producers in the Thunderhill area of the Swan Valley identified poor doing grazing cattle for many years. In 1953 Cunningham first documented copper deficiency due to excess levels of molybdenum occurring in the forages. Cunningham was the first to actually identify that Manitoba cattle suffered from a copper deficiency. In more recent works by Glen Findlay in a Masters thesis in 1962, and Manitoba Agriculture studies in 1974,75 and 76 by Drysdale and published in 1979 and work by Roy Boila have more recently defined a must larger area of trace mineral concern.

The forage studies carried out in the Northwest Region in the Parkland area of Manitoba by Manitoba Agriculture in 1974, 1975 and 1976 determined that severally other trace elements were also in short or marginal levels in the forages. The primary thrust has been however on the area of copper deficiency. A copper deficiency in cattle results in anemia, unthriftiness, poor growth, lameness and reduced milk and reproductive performance. a faded hair coat or a dry, rough hair coat which shows crescent shaped patterns of discoloration is common. Growth in calves is impaired and often shows instances of bone abnormalities and fractures. A primary copper deficiency is one that occurs due to low copper intake from the diet.

The occurrence of a secondary copper deficiency which is one caused by high levels of molybdenum and/or sulphates which reduce the availability and retention of dietary copper also occurs in the area. Animals on high molybdenum pastures or high sulphate waters will scour profusely, particularly with molybdenum, this occurs on lush spring pastures with legumes in the stand. In Manitoba, water quality is often a problem, in many deeper wells and occasionally even in shallow dugouts, conditions of high sulphate, high salts or a combination of both can occur. If this poor quality water is the only source available to the animals it results in a severe depletion of the trace mineral reserves in the animals. This can be particularly a problem in combination with the low copper levels that tend to occur in Manitoba. The primary and secondary copper deficiency problems are not isolated alone in Manitoba. They have been identified to quite an extent in British Columbia, some areas of Saskatchewan and Ontario.

The other trace minerals which tend to occur in less than optimal amounts are: zinc, manganese and selenium. These have been identified as low or marginal in many areas of the province. Effects of the high sulphate waters may result in severe imbalances in these trace elements as well.

Preliminary data from the Northwest Region in combination with other various analysis encountered in random studies in Manitoba indicates that the problem is not limited to the Northwest Region as was first thought. The complexity of the trace mineral picture in Manitoba is due in large to the geological history of Manitoba as shaped by the glaciers and the remnants of old Lake Agassiz.

Enclosed is the general geological map of Manitoba. The University of British Columbia, Dept. of Geology was instrumental in participating with Bob Drysdale in the studies that were done in the Northwest Region in 1974-75 and 76. Such characteristic geological features as the escarpment line running diagonally across from the central portion of the province northwest ward up to the Porcupine Mountains delineates many of the shorelines of the ancient lake. Associated with this escarpment is three mountain regions which are underlayen with high molybdenum shales which result in pockets of severely high molybdenum bearing forages around their peripheries. The increased use of alfalfa as a ruminant forage tends to aggravate the problem because legumes utilize molybdenum as part of the nitrofication process in the nodules on the roots. The other areas that quite often are very low and deficient in copper are the old beach ridges which occur along the escarpment line through much of the northwestern Manitoba and down into Central Manitoba. Most of the forage is often grown on sandy soils and have extremely low copper and trace mineral levels. These sandy soils occur in many parts of Manitoba. The location of these can be determined by a map of Manitoba Soil Types.

There is currently a trace mineral proposal being worked on under the Agrifoods Agreement in which a mapping of the forages found in Manitoba through analysis being carried out by the Manitoba Crop Insurance Feed Security Program. It is hopeful that this program will provide an overview of the entire Provincial situation and will serve as a source of guidelines for producers as to their potential trace mineral status.

One of the other major problems is that of water quality and the occurrence of poor quality waters in combination with either of two other factors, low trace mineral and/or high molybdenum levels in forage makes it very difficult to predict a particular situation for a farm without going into further extensive analysis of forage and water. There is a need for a specific mapping of problem waters in the Province and hopefully we can put together that picture along with the overall trace mineral forage analysis.

Water Resources Branch, Manitoba Dept of Natural Resources has maps of the Sand and Gravel Aquifers and Bedrock Aquifers which indicate where problem water may occur.

If producers have a specific problem that they feel is water related, their water services board technician can quite often give them an indication of where the high sulphate and problem waters occur in their specific districts in Manitoba.

In summary, the following observations have been made:

1. That copper levels in 75% of Manitoba feedstuffs maybe too low to meet animal requirements.
2. Molybdenum and sulphur levels may be a state of imbalance resulting in copper deficiency in over 65% of the grasses and 80% of the legumes.
3. The level of various trace minerals change in response to moisture conditions, soil formation and season.
4. The application of copper to soils does not improve copper levels in plants unless the soils is completely devoid of copper. Fertilizer with nitrogen or phosphorous increases the yield of grass and forage but does not necessarily increase the uptake of trace minerals.

5. The intake of sulphates through water and iron through the soil does have an effect on trace mineral balances.
6. Selenium levels are not well defined in Manitoba feedstuffs although selenium deficiencies are identified by the Veterinarians.

The determining of trace mineral status within a herd is not a simply and straight forward process. It requires analysis of forages, water quality and animal tissue samples to specifically identify the problems. There are several methods of supplementation such as use of trace mineral salts, high trace mineral packages, injectable copper and trace mineral elements as well as such things as boluses for the animals. However more research and work is required to specifically license injectable products in Canada and to determine the best methods of combatting trace mineral deficiency in animals grazing forages on pasture.

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The Frustration of Copper Supplementation

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Success in the cattle business is based on efficiency of production. One aspect of that production system is the nutritional status of the animal and the economics of improving its nutritional status in order to improve production. Optimizing the nutritional status of any animal is a matter of balancing intake of water, energy, protein, vitamins and minerals. An excess or deficiency in any one of these will result in inefficient use of the others. Many cattle producing areas of Manitoba have identified mineral supplementation as a major problem in achieving an optimum nutritional status for their herd.

Nutritionists and the feed industry categorize minerals as either macrominerals, which are required as g kg^{-1} quantities in the diet, or microminerals, required in mg kg^{-1} quantities. Table 1 provides a list of trace minerals required in beef cattle diets. Manitoba crops are low in cobalt and iodine and, therefore, it has become a standard practice to use cobalt-iodized salt for all livestock. Zinc, manganese, iron and selenium can be supplemented based on levels found in the diet. A trace mineralized salt is often used as insurance when diets are borderline adequate in these minerals. Identified deficiencies are often corrected through the use of specially prepared mineral mixes or premixes.

Manitoba crops have a wide range of concentrations for copper (Cu) and for the minerals that influence the animals' ability to use dietary Cu. As well, the water supply for cattle has been

found to contain a wide range of sulfates, also known to interfere with Cu metabolism. Therefore, the issue of Cu supplementation has become a source of frustration to the cattle producer, his feed supplier and the herd veterinarian. Genetic change in our beef cattle population, change in our feeding strategies and a greater awareness of trace minerals has increased the concern of Manitoba's cattle producers about the "when" and "how" of Cu supplementation.

Two major genetic changes have been identified as contributors to the increasing incidence of Cu deficiency. Firstly, great strides have been made in the last ten years for preweaning and post-weaning growth rates. For example, ROP data shows that the 205 day adjusted weaning weight in 1979 was 221.8 kg. The estimate for adjusted weaning weight in 1989 is 244.0 kg. This increase in growth rate represents an 11% increase in the daily Cu requirements of the calves. The second genetic change was the introduction of Simmental cattle into our herds. Currently, it is estimated that 20-25% of Manitoba's beef cattle contain some Simmental blood. It is now established that Simmental cattle are more susceptible to Cu deficiency because they excrete more Cu in their bile. Consequently, diets that were borderline Cu-adequate for the traditional cattle breeds on a particular farm are resulting in copper deficiency problems when Simmental is incorporated into the breeding program. Susceptibility to Cu deficiency may vary within breeds as well. Several researchers have reported fairly high heritability estimates (0.44 to 0.65) for plasma Cu concentrations. It may be that cattle producers in Cu deficient areas should be considering breed and plasma Cu levels of the bulls they bring into their herds.

Changes in feeding or management strategies also have contributed to the increased incidence of Cu deficiency. For example,

there has been a shift in the calving period from April-June in 1979 to February-April in the 1990's. In general, calves are born with liver copper reserves that are used up during the suckling period because milk provides virtually no Cu. These calves start to consume significant amounts of forage by the time they are 3-4 months of age and, generally, start to recover body Cu reserves at that time. Pastures low in Cu and high in molybdenum (Mo), a mineral that interferes with dietary Cu absorption, do not allow this recovery to take place. In the traditional calving schedule, late-born calves would rely on pasture forage for a matter of 2 or 3 months before being moved to over-wintering rations. Early-born calves, however, are exposed to these copper deficiency conditions for 4 to 5 months. Consequently, producers who have not changed pastures, have not changed the over-wintering diets but have moved up the calving time, can suddenly be faced with losses due to Cu deficiency. Another consequence of this change may be reduced conception rates on the high Mo pastures because the highest levels of available Mo are found during the period of rapid pasture growth. There is increasing evidence that high dietary Mo affects reproductive hormones in females and males, the end result being reduced fertility. Shifting the breeding period from July - August to May-June would expose animals to high Mo levels during a critical time of the reproductive cycle. Again, this is a situation in which the producer has not changed pasture or winter feed supplies, but suddenly is faced with a Cu-Mo problem.

What do we know about Cu for cattle? Firstly, liver Cu levels are the most reliable indicator of an animal's Cu status. Table 2

provides concentration of Cu in the liver of cows and newborn calves defined as having either a normal Cu status or a Cu deficient status. In Western Canada, it is important for calves to be born with 250-300 mg Cu kg^{-1} DM to survive the suckling period. In order for calves to achieve this liver concentration, cows should be coming off pasture with liver Cu levels of approximately 50 mg kg^{-1} DM. Plasma or serum Cu levels are reliable only when concentrations are less than 50 mg dL^{-1} (0.5 mg L^{-1}, 0.5 mg ml^{-1}), in which case Cu deficiency is established.

Secondly, we know that there are three basic situations that can lead to a Cu problem in Manitoba beef cattle herds. Cattle may simply be consuming a diet that is low in Cu. Various studies at the University of Manitoba have identified that the concentration of Cu is less than 10 mg kg^{-1} DM for 50% of the forage grown in the province. A second situation resulting in a deficiency is excessive levels of Mo (> 3 mg kg^{-1} DM) in forage. Approximately 50% of forages grown in Western Manitoba would fall into this category. Mo interacts with sulfur (S) as it passes through the rumen and is converted to a series of compounds referred to as thiomolybdates. These thiomolybdate compounds associate with particles in the rumen fluid (bacteria, protozoa and plant material) to form strong chelators which have a strong attraction to Cu and will not release the Cu as it passes through the acid conditions of the abdomen and small intestine. The end result is that Cu consumed by the animal is excreted in the feces. A small portionof the thiomolybdates are absorbed into the animal's blood stream. This reduces the efficiency of Cu metabolism in the animal and is also one of the main reasons why plasma or serum Cu concentrations are not a reliable indicator of animal Cu status. The third si-

tuation in which Cu deficiencies have been observed to occur is high sulfate (SO_4) levels in water and, to a lesser extent, high sulfur levels in forage. In general, water containing more than 600 mg L^{-1} sulfate or forage containing more than 2 g kg^{-1} DM sulfur are considered to contribute to Cu deficiency. Rumen microbes need approximately 0.10 g kg^{-1} DM or 0.1% sulfur in the diet for optimum fiber degradation and protein production. Dietary sulfur concentration is increased the equivalent of 0.10% for every 1000 mg SO_4 L^{-1} in water. Even when diet Mo concentrations are low, sulfur can have a negative effect on Cu absorption. Sulfur containing compounds are converted to sulfide (S^{2-}) in the rumen. Copper released in acidic conditions of the abomasum becomes bound to S^{2-} to form cupric sulfide which is excreted in the feces. We often observe plasma Cu levels of animals grazing summer pasture to be higher than for animals grazing lush spring pasture. It is suggested that the highly soluble proteins of rapidly growing grass result in higher S^{2-} production in the rumen, decreasing availability of dietary Cu.

Disease syndromes associated with Cu deficiency in cattle include anemia; impaired leukocyte function which results in reduced immuno-competence: swelling of joints and increased brittleness of bones due to defective osteoblast functions; loss of hair color, and decreased growth rate. Although Cu responsive infertility has been reported in dairy cows, the most frequent cause of poor fertility in Cu-deficient animals occurs if animals are consuming high levels of Mo. Excess

MO in the diet has been associated with delays in puberty, disruption in estrus activity and lowered conception rates in cattle. It has also been associated with reduced sperm numbers in rams but has not been evaluated for bulls. Polioencephalomalacia, also referred to as Bl deficiency, has been observed in animals fed low Cu and/or high S diets although the exact mechanism is not known. The incidence of abomasal ulcers accompanied by sudden death increases for calves that are Cu deficient. Several theories have been presented about the relationship between animal Cu status and this syndrome.

Three problems Manitoba cattle producers are faced with when planning a Cu supplementation program for their herd are: the amount of Cu animals should be given; the high cost of some Cu supplements and the lack of availability of most Cu supplements. Types of copper supplements available and recommended supplementation rates will be presented.

Table 1. Suggested requirements and dietary allowances for trace minerals in the diet of beef cattle (NRC 1984)

Mineral	Requirement (mg/kg DM)	Dietary Allowance (mg/kg DM)
Cobalt	0.07 - 0.11	0.1
Iodine	0.2 - 2.0	0.5
Zinc	20 - 40	50
Manganese	20 - 50	50
Iron	50 - 100	50*
Selenium	0.05 - 0.30	0.2
Copper	4 - 10	10**

*Dietary allowance is 100 mg/kg for growing calves.

**Requirement for Cu when molybdenum is less than 2.0 mg/kg DM and sulfur is less than 3.0 g/kg DM

Table 2. Concentration of Cu (mg kg^{-1} DM) in the liver of normal and Cu-deficient cattle

	Animal Cu Status	
	Normal	Cu-deficient
Newborn calf	381	55
Cow	200	11.5

TRACE MINERALS - A VET'S PERSPECTIVE

Presented by Dr. James Dyck

As said earlier, I grew up in Holland a little way from here and had never heard of Swan River, let alone copper and/or selenium deficiency. As a veterinary student in the early 1970's, I helped on the trace mineral study that Bob Drysdale and his group did in the northwest region (I eartagged the animals). It was quite an eye-opener, as I saw the extent and signs of copper deficiency in the Swan Valley.

This was my first taste of trace mineral deficiencies and I was quite intrigued. Copper deficiency had been recorded since the 1910's up in the Swan River area especially around Thunderhill. Selenium deficiency was predicted in the 1800's, due to the soil type. At the Western College of Veterinary Medicine, the topic was covered briefly but more importance was placed on toxicity.

Why are we more concerned with trace minerals now than 25 years ago? There are a variety of reasons that come to mind.

Firstly, our cow and calf size is larger. In the years that I have been in the Swan Valley, average cow size has increased by 250 lbs. Calves are now being born from these cows at 120-130 lbs. unassisted and raring to go. Nutrition levels have to be much more exact to get these big healthy calves. We are now weaning 700-800 lb. calves. Looking over the ROP records for 1970-1989, we see 150 lb. increases in weaning (200 day weights) and 300 lb. increases in yearling weights.

We have changed our feeding dramatically from straw and grain or wild hay to legume - grass and grain. Energy and protein are no longer the major limiting factors. Minerals rise to the forefront as holdups of production.

Along with the change in feed, we have changed our management of pastures and grasslands. We fertilize much heavier and, as pointed out, sulphur in fertilizer will affect both selenium and copper availability. We are also using a lot more tame pastures.

As veterinarians, we recognize these problems and are able to address them. We have the technology to prove or disprove a deficiency, and monitor levels more economically and faster. When I started practising we wondered, but the technology was costly or non-existent to prove it, and results were slow in coming back.

When I started work in Swan River, the problem became almost a daily business as up here all areas are deficient in copper to some degree. A standard treatment for copper deficiency was one or two lbs. of copper sulfate in the mineral or injecting with copper when it was available. But this left a lot of holes and cattle and calves on treatment were still showing signs of deficiency.

I was involved with several studies in this area looking at copper and selenium. The first looked at copper and selenium in fetuses and newborn calves and feed levels. All the tested feed samples showed low copper levels and adequate selenium levels in the feed. Liver values were normal for copper

except for one sample and these animals were supplemented with copper. Selenium liver values were in the normal range but interestingly, one of the most severe white muscle cases occurred on the farm where feed and liver levels appeared adequate.

The second looked at cow-calf pairs on pasture and blood selenium levels through the summer. Bloods were drawn in June, July, and September. Two herds showed a decrease in selenium levels in the June to July testing but all showed an increase in levels in the fall, although all were in the normal ranges. Did the lush spring pastures lower blood levels on the marginally deficient pastures, and higher fall pasture levels are reflected in the fall values? I'm not sure.

Scours in little calves gave the biggest day-to-day concern, as response to treatment was variable. Dr. Leach found that iron dextran (pig iron) would have an effect on this problem. It did not prevent the scours in copper deficient calves, but the calves did not get as sick and were much more treatable when they were given a treatment at birth. It has now become a routine procedure with many of our producers.

Selenium problems were more subtle. Selenium seems to be the hidden denominator in a lot of little problems.

Reproductively, I saw it first in dairy herds. At 30 days post calving the bovine uterus should be almost back to normal. On some dairy farms, I was finding 40-50% of the cows with abnormal uteruses on 30 day checks. These cows were calving normally and cleaning normally, with no history of

reproductive problems at calving. Injecting with Se/Vit E dramatically dropped the percentage of abnormal uteruses found to less than 10%. I started to use 10% as a rule of thumb in our beef herds. If it was above this level it was time to look at nutrition. Often Vit E/Se were in short supply and rectifying the problem would result in a lot fewer retained placentas.

I saw a lot of crooked legs and weak calves in my early years in Swan River. I had begun to dread seeing big calves (100 lbs. plus). They were hard to get out of cows except by cesarean section, it seemed. Once you had them out, they were weak, slow to get going and a lot would have front legs that liked to buckle under them. Treatment with Vit E/Se on these calves would straighten up these legs in half the time with no casts or splints.

But what do I see that makes me start thinking of these trace minerals?

I will start with copper, as it seems to be our major concern. We have areas up here that are just short of copper, period, and that varies from year to year. The faster the growth (i.e. spring and wet weather), the lower the copper levels. Copper: Molybdenum of less than 5:1 indicates a problem. Sulphur higher than 3.0 g per kg feed is considered a problem. Legume versus hay can result in higher Molybdenum levels and a secondary copper deficiency.

We still see calves like this, thin, poor hair colour,

and a little diarrhea. These calves are poor doers and are the first to get sick in bad weather. We will see enlarged growth plates on the legs and reports tell us that legs will break easier. Often we see it in calves when we treat for pneumonia or scours. A minor problem does not clear up like it should, and on checking copper levels we find deficiencies. Treating with copper results in proper response to treatment.

With severely deficient cows we see the diarrhea, poor doers, and poor conception with strung out calving. Their calves tend to be sickly and response to treatment is poor. Less severe deficiency results in poor pregnancy rates.

Selenium is much more subtle, other than white muscle disease where you see a stiff calf, or down animal, its effects can be seen as a lot of other things. Now I will talk about selenium, but selenium and Vitamin E interact so that a shortage of one will interact and lower the availability of the other. Conversely, treating with one will help alleviate to some degree, the deficiency of the other.

Reproductively, we were seeing more retained placentas after calving than is normal. I consider more than 10% retained placentas at 8 hours time a problem and should be looked into. But a lot of things can cause retained placentas. Nutritionally all items should be scanned, energy, protein, and minerals. When pregnancy testing, these Selenium deficient herds, the open cows did not seem to have normal uteruses, they were bigger and abnormal feeling. I found good growthy, exotic heifers were cycling well but conception was poor. When we started injecting Vitamin E-Selenium, conception rates were much improved.

As Selenium is important in muscles (any kind of muscle) stress or strain can be exaggerated with a selenium deficiency. Extra movement or handling can bring on all kinds of limping and apparent sprains. Very excitable animals allowed to get really worked up and then chased have been known to go down and die shortly thereafter. The heart is a muscle and it will not handle the stress.

Selenium is also involved in the body in the immune system. Selenium-Vit E is necessary to fight disease and when it is in short supply, the body is unable to build up adequate defense throughout the immune system, once exposed to an infection. Reports of Vit-E selenium shortages resulting in higher incidences of mastitis and infected quarters are being filed. Poor response to antibiotics when a pneumonia outbreak occurred in a feedlot is a personal experience. The calves responded poorly until Vit E-Selenium was added to the treatment. When selenium blood levels were checked I found selenium deficient calves.

As I mentioned, when I started to practise in Swan River, I did not enjoy seeing 100 pound and larger calves. They had crooked legs, were weak and just slow to get going. When selenium supplementation was started, we find these problems disappeared. Now our big calves are up and going, as well as our smaller ones.

I have seen some Zinc responsive problems, mainly cracked feet and real hard feet to trim (the hoof is hard and really brittle). Once zinc supplementation is started we get rid of these cracks and the feet soften.

What do I want when we are looking to confirm a trace mineral deficiency?

When we started looking at copper problems, we took hair samples and sent feed samples for Copper and Molybdenum. We knew that hair samples reflected levels weeks and months earlier. Feed samples were slow to get back. On top of this, it does not take into account any interactions except Molybdenum. Now I draw blood samples and in the case of a herd problem, I do three or four age groups (mature cows, replacement heifers, feeder steers, and calves), depending what is on the farm. As ration varies from group to group, they could vary greatly in their status. I try to do anywhere from 5-8 head in each group. The Manitoba Provincial Vet Lab has the capability to do blood copper tests quickly, with a turnaround time of 2-3 days, at a very reasonable cost. You can get a picture quite quickly on the herd.

I collect liver samples every chance I can. The liver is like the well with the blood the outlet. You know when the well is very low, when the outlet shows low levels, but if it is just about empty, only looking in the well will tell you that. And that is the liver. I routinely ask for copper and selenium on all aborted fetuses and postmortem submissions. If I get a chance, I go to the abattoir and collect livers from produce cattle, as it will show what is happening in those herds, either as feeders or cows. Results are a little longer getting back but provide a good check on where your supplement levels are at.

With deficiences, diagnosis has become quite a bit easier and cheaper in the last few years. In the early 80's, I did several studies looking at feed, blood, and liver levels. With samples of Selenium in the range of $15-$25, it ate money up quite quickly. Also, it took anywhere from 6 weeks and up to get results. Okay for a study but for tying to ascertain levels on a routine level, it was not effective. In the last couple of years, the Provincial Lab had a machine available to do blood seleniums and recently, got it functional. I have almost stopped doing feed analysis with interactions, normal values seem to have little meaning. I have had cattle on feed with selenium values well within the normal range and saw classic white muscle disease. Again, I make use of any liver samples I can get as it is the best way for evaluating the body stores.

Zinc also can be blood sampled for and our local Provincial Lab will analyze it. A lot of our treatment levels are on an individual basis as every area has different degrees and causes of deficiency.

Copper recommendation was fairly standard with 1-2% Copper Sulphate in mineral or salt. But there is a lot of difference between a ½ oz. intake per day and a 2 oz intake per day of mineral. Also we know that all animals do not take in an equal amount of mineral. I have seen Copper deficient. Now I recommend on average an intake of 1 lb. of Copper Sulphate to 90 beef cows per week. It is easier and more accurate. Calves we allow for weight and add a little extra.

At birth, we routinely use 5 cc of Iron Dextran. In severe areas, we will also give injectable copper to the calves at the same time. You will get swellings but these disappear in 2-3 weeks, with small lumps left occasionally. Once calves are 3 months old we will reinject our calves before hitting pasture, if we are on severely deficient pasture. Milk is a poor source of copper and will not increase in values above normal if the cows are over supplemented. We can and will re-inject in the fall also. The product that we use will last only 35 days in severely deficient areas, as reported by some veterinarians but longer if feed values are higher.

Top dressing pastures with copper sulphate has been tried in the past and it did significantly raise the copper levels but as there is a time when the levels are in the toxic range, cattle have to be held off pasture. The copper sulphate adhered to the leaves and needed to be cleared by time or a heavy rain. In retrospect, it has never caught on up here, although copper deficiency in cereal grains has been diagnosed up here and copper sulphate added to the soil.

Boluses of copper, selenium and cobalt have been developed that are incorporated into glass. A slow release of all the minerals results in a possible long-term treatment (12-18 months) is possible. They are being used in Australia and New Zealand but they are not available in Canada commercially yet. I think this is one of the most promising treatment methods of the future.

With selenium we inject the calves at birth with a Vit E-SE combination (6 mg of selenium and 277 IU of Vitamin E) at twice the recommended levels. We will reinject at the time when they hit pasture if early calves (Jan.-Feb.) and in the fall. We have a couple of severely deficient pastures and we have to reinject on pasture. Cows we inject with Vit E-SE (35 mg selenium, 1600 IU Vit E) in the fall and feeding Sel - Vit E through the winter period. I try to get about 2.5 - 3 mg per day of selenium into the cows and calves.

Top dressing of pastures has been attempted with Selenium selenite and has given an increase in plant levels of selenium but one must be careful as plant levels fall into the toxic range after top dressing for a while and cattle may be held off pasture for some time. A lot of selenium is required to make any noticeable rise to plant levels. A high percentage is tied up in the soil and this makes it expensive. As I have suggested, boluses have been tried with good success elsewhere but are not available here.

To date, we have found that the trace mineral salt and minerals seem to have enough zinc to alleviate the zinc problems we see.

No talk should be complete without a discussion on toxicity. Cattle seem to be quite tolerant to high copper levels but high levels of copper sulphate can cause intestinal irregularities. Chronic poisonings have occurred. Remember also that sheep are especially susceptible to copper poisoning and access by sheep to copper sulphate has to be regulated. Horror stories abound of sheep getting into high copper mixes. Selenium is well known to cause chronic problems in cattle and, as such, pay attention to dosages. The old rule I often hear "If 1 pound is good, 2 pounds are better" could cause a lot of grief.